Effects of cyanotoxins on California wildlife and the land-sea connection

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Key cyanotoxins of California fresh & brackishwater systems

- **“Hepatotoxins”:** Microcystin, nodularin, others…
- **Neurotoxins:** Anatoxins, saxitoxin, others…
- **Pro-Inflammatory:** LPS, others…

- At present, what we know is far less than what we don’t know
- Impact assessment hindered by communication obstacles, limited testing, limited understanding
Why? Diverse routes & mechanisms of toxicity = diverse disease!

- **Routes:** Oral, inhalation, aspiration, dermal, IV, IP

- **Mechanisms:** Neurotoxicity, neurodegeneration, anticholinesterase, phosphatase inhibition, necrosis, apoptosis, oxidation, sensitization, inflammation, immunosuppression?, O2 depletion, food web alteration, biomagnification, facilitation of botulism outbreaks?, synergy with other toxins/pathogens?, DNA damage, teratogen, tumor promotor
Additional challenges to estimating cyanotoxin impacts:

- DVMs & MDs: Minimal training re cyanotoxins!
- Taught that there is no Tx for this condition
- Existing descriptions: Experimental (rodents) & individual acute, severe cases
- Diverse toxins + varying production/exposure/duration = Spectrum of disease
- Result: Most cases are missed, including by top-notch veterinarians & doctors! Few cases examined or tested Fewer published

= Disease prevalence greatly underestimated & numerous animals die without Tx
Monterey Bay: At least 4 sets of unexplained dog illness/death from acute liver Dz following water contact since 2007, including 2 dogs owned by DVMs!

No dogs examined postmortem, none tested for cyanos!
Detection is highest where surveillance is greatest: Pets, livestock, T & E spp., mortality events, aquaculture, mariculture
Selected California counties with suspected or confirmed deaths due to cyanotoxins (mammals & birds)

#1 Microcystin

#2 Anatoxin

Huge potential for food web transfer → Unrecognized impacts
What can sea otters teach us about microcystin?

High-risk habits in a polluted world

- Nearshore-dwellers w/ strong site fidelity
- Often congregate near bays, harbors, river mouths
- Consume 25-30% of body weight/day
- Primary prey = filter-feeding inverts (clams, mussels, worms.....)
- Filter-feeders remove both biological and chemical pollutants from contaminated water
Many sea otters die following exposure to land-based pollutants.
2007: We begin noticing stranded, bright yellow otters

Icterus (jaundice): Oral mucosa and rib cartilage

Normal sea otter liver

Liver from sick sea otter

Normal liver (microscopic view)

Liver from sick otter (microscopic view)
Beginning the investigation at the Pajaro River mouth

Pinto Lake Park

- Launch Ramp
- Picnic Areas
- Boat rentals
- Fishing

X- Sick sea otter

Monterey Bay

~8.5 km
Stepwise sampling from Pinto Lake to the ocean, fall 2007

- Pinto Lake surface scum: 2,900 ppm
- Corralitos Creek also +
- Pajaro River + within 1 km of the ocean
Current case distribution
How are sea otters getting exposed?

- **Rapid lysis:** *Microcystis* nearly 100% lysed >24h in seawater

- **Environmental persistence:** Freshwater microcystins stable in seawater (still easily detectable after 21d)

- **Endemic & commercial spp are excellent bioaccumulators:** clams, mussels, oysters & snails > crabs. Bivalve GI up to 107x > [microcystin] than seawater (up to 1,324 ppb ww)

- **Slow depuration:** Bivalve GI tissues strongly + 2 weeks PE, despite exposure to clean seawater >4d PE
Although microcystin appears to be originating from freshwater sources in CA, intoxication is pretty common in sea otters

- 31 cases, 1999-2011: First report in any marine mammal
- All LCMS/MS + (Limited testing)
- Additional LCMS/MS- suspects (bound MC?, other cyanotoxin?)
- Equal proportion of males, females
- All age classes (~75% adults)
Cyanotoxin cases may be increasing through time in CA & can present any time of the year.

Sea otters ➔ dogs, cattle, horses, fish, birds, goats
Not all affected animals die acutely

- 16 otters stranded alive
- 6 lived 4-70 days post-stranding \( \rightarrow \) Still MC+!
  (Prolonged toxin retention D/T enterohep. recirc.)
- Clinical signs often vague (ADR) \( \rightarrow \) Euthanized
- Few recognized clinically as microcystin cases
- Suspect foods: Clams, mussels, crabs, worms, milk
It’s not only about the liver!
Phosphatases are present in most tissues!
(Additional targets: Brain, heart, kidney, BVs, etc.)

Otter that died from microcystin intoxication

Hemorrhage, internal heart wall

Hemorrhage & Vacuolation, heart muscle
Spectrum of disease (easy to miss), synergy with other toxins/ pathogens?

• < 1/2 were icteric (yellow)
• Nearly 1/3 had normal looking livers (grossly)
• 58% had evidence of abnormal blood clotting
• > 1/2 had gross cardiac lesions
• ~2/3 had gross brain lesions
• ~2/3 had significant bacterial infection
• Synergy with other biotoxins? (eg. cardiac Dz)
How can we optimize mitigation of cyanobacterial impacts in CA?

- Improved awareness (DVMs, MDs & the public)
- Organized reporting & mapping of suspect cases
  → ID “High risk areas” for focused mitigation
- Match cyano testing & pathology
- ↑ test availability, ↓ cost, test validation & standardization.
- Focused research on food web dynamics, high-risk spp., human health risks, disease spectrum

SPATT: Bloom dynamics, locations, potential source(s), etc.

ANIMALS/ PEOPLE: ID high risk areas, food web dynamics, human health risks, case recognition, treatment, public notification
Benefits

- ↓ Health risks: Animals & humans
- Fewer animal deaths
- Problem-focused mitigation
- Improved water quality & food safety
- Ecosystem-wide wildlife benefits
- Conservation of T&E spp.
- “Take” of wildlife & T&E spp.-Illegal under state & federal law, including water pollution
- People care about this!
Thank you!

CDFG, MBA, USGS, UCSC, UCD, TMMC, USFWS, TMMC, California State Water Quality Control Board, California Central Coast Water Quality Control Board, CDFG Water Pollution Control Laboratory, City of Watsonville, Applied Marine Science, CDPH

(Photo credit: Randy Wilder, Monterey Bay Aquarium)